EDITORIALS

Contribution of Echocardiography to the Understanding of the Pathophysiology of Cardiac Tamponade

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Echocardiography has become the tool of choice for the diagnosis of pericardial effusion. Its presence is readily identified by M-mode echocardiography. Two-dimensional echocardiography provides a clearer view of the extent of the echo-free space that indicates the presence of a pericardial effusion and is useful in excluding other, less common causes of an echo-free space.

In addition to identifying the presence of abnormal pericardial collections, the echocardiogram has been found to be a useful adjunct in the diagnosis of the potential hemodynamic consequence of pericardial effusion—cardiac tamponade. Cardiac tamponade is a clinical and hemodynamic diagnosis, not an anatomic one. Depending on the size of the heart, the rapidity of intrapericardial fluid accumulation and the distensibility of the pericardium, tamponade may occur with small or large effusions. Because the echocardiogram provides anatomic rather than hemodynamic information, it would seem unlikely that this tool would be useful in establishing a hemodynamic diagnosis. However, correlation of a series of echocardiographic observations with clinical and experimental studies has overcome this apparent limitation and, in the process, has yielded new insights into the pathophysiology of tamponade and such related matters as the basis for paradoxical pulse and electrical alternans.

Phasic respiratory changes in cardiac tamponade (paradoxical pulse). Normally, inspiration causes an augmentation of peripheral venous return that results in enlargement of the right ventricle. In cardiac tamponade, the pericardium can no longer distend; the increase in right ventricular volume must occur at the expense of left ventricular volume (1). Inspiratory pulmonary intravascular pooling in the face of a reduced cardiac output may contribute to the phasic changes in ventricular volume and output in patients with cardiac tamponade; however, the magnitude of this contribution is unclear.

Echocardiographic studies by D'Cruz (2) and Settle (3) and their co-workers identified the presence of phasic respiratory changes in left ventricular dimensions and mitral valve motion in patients with cardiac tamponade. Left ventricular volume and mitral valve excursion were noted to diminish during inspiration. Right ventricular dimensions increased with inspiration and thus the interventricular septum "shifted" toward the left ventricle. These abnormalities resolved after removal of pericardial fluid. These observations were reinforced and expanded by other investigators (4–6). Schiller and Botvinick (7) demonstrated that the echocardiographically determined right ventricular internal dimension was decreased in patients with cardiac tamponade as compared with the size of the right ventricle after removal of pericardial fluid.

Paradoxical pulse in absence of cardiac tamponade. Unfortunately, the phasic respiratory changes in ventricular size and mitral valve excursion are not specific for cardiac tamponade. We have shown that these changes may occur in a variety of conditions associated with other causes of paradoxical pulse (8,9). It has long been recognized that any condition that causes an exaggerated decrease in intrapleural pressure will produce a similarly exaggerated decrease in arterial pressure on inspiration. The echocardiographic observations of this phenomenon run contrary to the opinion that the decrease in systemic pressure is simply the result of the transmission of the lower intrapleural pressure to the aorta. It has been demonstrated that the magnitude of paradoxical pulse is proportional to the inspiratory decrease in left ventricular dimension, diastolic volume and stroke volume (9). An inspiratory decrease of left ventricular stroke volume is the cause of paradoxical pulse, whether due to cardiac tamponade or respiratory distress (10). In one extreme example of this phenomenon, a patient with acute pulmonary embolization had such marked inspiratory

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reduction of left ventricular stroke volume that the arterial pressure recording showed no deflection during inspiration, while the echocardiogram demonstrated failure of the aortic valve to open. This patient had no pericardial effusion and his paradoxical pulse was clearly related to inspiratory reduction in flow across the aortic valve rather than to inspiratory decompression of the aorta (8).

A correlation between echocardiographically determined valve motion and transvalvular flow has been well documented in open chest dogs (11). In cardiac tamponade and airway obstruction, the flow across the mitral and aortic valves is reduced during inspiration; therefore, the echocardiogram shows an inspiratory reduction of mitral and aortic valve excursion and decreased mitral valve EF slope.

Absence of paradoxical pulse in cardiac tamponade. Paradoxical pulse may be absent when chest wall trauma or neuromuscular disease results in an inability to develop the normal respiratory changes in intrathoracic pressure. When patients are on a positive pressure respirator, there is reversal of the normal, phasic intrathoracic pressures. The resulting inspiratory elevation of intrathoracic and aortic pressures will prevent the development of paradoxical pulse (12). These effects are important to recognize because cardiac tamponade may develop in patients who have undergone cardiac surgery and who are still receiving ventilatory support. Paradoxical pulse is also uncommon with cardiac tamponade in the setting of severe aortic regurgitation. When right and left ventricular volumes do not undergo respiratory variation, as in patients with a large atrial septal defect, the development of cardiac tamponade is not accompanied by paradoxical pulse. Echocardiographic study of these patients shows no respiratory variation in chamber size or valve motion (13).

Other echocardiographic signs of cardiac tamponade. Shina et al. (14) recently reported the echocardiographic findings of diastolic posterior motion of the anterior right ventricular wall as a sign of impending cardiac tamponade. This finding was confirmed by Armstrong et al. (15), utilizing both M-mode and two-dimensional echocardiography. A review of our cases of cardiac tamponade revealed this sign to be a consistent finding. The pressure in the pericardium increases during diastole as a result of the increase in left and right ventricular volume. Because the thin-walled right ventricle is the more compressible of the ventricles, it sustains the brunt of the elevated pericardial pressure; diastolic right ventricular compression is the result (16).

Vignola et al. (6) observed a systolic notch on the right ventricular epicardial echogram in four patients with cardiac tamponade. The notch occurred 0.04 ± 0.01 second after the QRS complex and was not seen in patients with a large pericardial effusion but without cardiac tamponade. The reason for this finding is not well understood.

Electrical alternans. Electrical alternans is seen in patients with a large pericardial effusion. Numerous echocardiographic studies have demonstrated that this beat to beat alternation is seen in association with changes in the distance between the chest wall and the heart (17,18). A pendulum-like motion of the heart within the enlarged pericardial space has been the common explanation for electrical alternans, the resulting change in cardiac position producing a shift in electrical axis that is seen as electrical alternans. However, there has been no satisfactory explanation for the alternation of cardiac position on successive beats. Removal of small amounts of pericardial fluid has been shown to eliminate electrical alternans (17,19), even though the residual volume within the pericardium was so large as to make it unlikely that the motion of the heart would be altered by this removal. Electrical alternans may therefore reflect a hemodynamic abnormality. This supposition is supported by reports of patients with pericardial effusion with electrical alternans in whom the electrocardiographic abnormality coexisted with major clinical evidence of hemodynamic derangement (17,19,20).

Cardiac tamponade remains a clinical entity and no single echocardiographic sign is 100% specific or sensitive for this condition; however, echocardiography can aid in its diagnosis and improve our understanding of its pathophysiology.

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